Team Approach: Treatment and Rehabilitation of Patients with Spinal Cord Injury Resulting in Tetraplegia

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Abstract

» Cervical spinal cord injuries result in limited upper extremity function and often lead to loss of independence.

» Tendon and nerve transfers have been shown to reliably improve upper extremity function.

» Most patients with injuries at or caudad to the C6 level can become independent through tendon and nerve transfers.

» Nerve transfers have expanded the reconstructive options beyond what was available with just tendon transfers and often can allow the patient to obtain 1 or 2 more functions than previously possible.

» Patients and physiatrists can be reticent to consider upper limb surgery because they fear compromising long-term function when a cure for spinal cord injury is discovered.

Clinical Scenario

A 16-year-old boy presented to a spinal cord injury treatment center for evaluation 6 months after diving headfirst into a shallow pool and sustaining a complete spinal cord injury at American Spinal Injury Association (ASIA) spinal level C6[1]. He had received intravenous methylprednisolone within 8 hours after the injury and had undergone a stabilization and decompression of a C4-level spinal cord injury within 24 hours after the injury. He had a tracheostomy scar in the midline of the neck. He sustained no other injuries but did have to be resuscitated at the scene after nearly drowning. Examination revealed a well-nourished male who used a motorized wheelchair, with 99% oxygen saturation in room air. Shoulder abduction, internal and external rotation, and horizontal abduction were classified as grade 5/5 according to the system of the Medical Research Council (MRC)[2]. Elbow flexion and brachioradialis strength were classified as 5/5. Elbow extension was classified as 0/5, with no triceps tendon reflex. Wrist extension was classified as 3/5 to 4/5 on the right and as 5/5 on the left. There was no distal function, but the finger flexor and intrinsic muscles were spastic with hyperactive reflexes and an intrinsic plus posture of the hand. The International Classification for Surgery of the Hand in Tetraplegia (ICSHT) score[3] was therefore 2 on the right and 3 on the left, with a narrow zone of injury at C6-C7.

After extensive conversations with the patient and the family as well as an evaluation by the occupational therapist, a surgical plan was devised to restore triceps, pinch, grasp, and finger extension bilaterally. Seven months after the injury, with 2 surgical teams operating simultaneously to minimize anesthesia time, the patient underwent a bilateral transfer of the

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posterior deltoid branch of the axillary nerve to the common triceps branch of the radial nerve, a right transfer of the nerve to the supinator to the anterior interosseous nerve, and a left transfer of the nerve to the supinator to the posterior interosseous nerve.

Twelve months after surgery, the right side did not have elbow extension but did have active composite pinch and grasp. The left side had active triceps strength (3/5) and active finger extension strength (3/5). A second procedure was performed 20 months after the injury to restore triceps function on the right (with a biceps-to-triceps tendon transfer), finger and thumb extension on the right (with a brachioradialis-to-extensor pollicis longus and extensor digitorum communis tendon transfer), grasp on the left (with an extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer), pinch on the left (with a brachioradialis-to-flexor pollicis longus tendon transfer), and correction of the Froment sign[4] (with a bilateral split flexor pollicis longus tendon transfer).

Twelve months after the second procedure, <3 years after the initial injury, the patient had bilateral active elbow extension (4/5 on the right and 3/5 on the left), bilateral pinch and grasp (3/5 on the right and 4/5 on the left), bilateral finger extension (4/5 on the right and 3/5 on the left), and unilateral supination (absent on the right but 5/5 on left). He was able to self-catheterize and self-administer a suppository for bladder and bowel function, respectively. He could weight-shift in bed and could get in and out of the wheelchair on his own. The motorized wheelchair was replaced by a manual wheelchair with power-assisted wheels. He was able to attend college on his own in a wheelchair-accessible dormitory.

Introduction

The care of those who suffer from a spinal cord injury has improved dramatically since the end of the Second World War. Specifically, advances in acute management have allowed patients to survive what had been a predominantly fatal injury, advances in acute and chronic rehabilitation and medical care have improved the ability to minimize complications and extend life expectancy, and advances in tendon and nerve transfer surgery have improved the ability to restore vital upper limb functions. Approximately 54 people per million sustain injuries to the spinal cord each year (corresponding with 17,000 new cases per year), with about half occurring at the cervical spine and 14% resulting in ventilator dependence[5].

Despite a decreasing incidence of such injuries, improved care has led to a rising prevalence, as more patients survive both the initial injury and the long-term sequelae. About 300,000 Americans now live with some residual paralysis resulting from a spinal cord injury. Most of them were young men when the injury occurred, and many require lifelong care to complete activities of daily living (ADLs) such as feeding, dressing, bathing, perineal care, and mobility. Maintenance cost estimates of care range from $40,000 to $185,000 per year per patient, depending on the severity and level of the injury[5].

Injuries range from complete to incomplete, with multiple incomplete injury patterns of varying prognosis. Although the ASIA Impairment Scale (AIS) is most commonly used to describe the severity of the injury to the spinal cord, the ICSHT classification system is more relevant for upper limb reconstruction (Table I)[3].

Acute care, from the management of the patient at the site of injury to transportation and spinal stabilization, focuses on minimizing further trauma to the spinal cord. Rehabilitation following stabilization of the injury focuses not only on minimizing complications such as pneumonia, urosepsis, decubiti, and autonomic dysreflexia but also on maintaining joint mobility and muscle tone. The importance of counseling and family support in the difficult time following an injury cannot be overstated. Early introduction to a hand surgeon who is familiar with reconstructive options for patients with tetraplegia has been shown to increase the likelihood that the patient will consider surgical options after maximum spontaneous recovery has been achieved[6], typically between 6 and 12 months in cases of complete spinal cord injuries.

Minimizing complications in the long term requires education of (and participation by) family members and the patient's support network, establishment of required professional home care, and, most importantly, optimization of the patient's own function. For patients with diaphragmatic paralysis, restoration of negative-pressure ventilation with a phrenic nerve pacer is paramount. For those with ICSHT grade-1 involvement and above, restoration of some hand function is possible with a combination of nerve and tendon transfers.

Acute Injury Management

The initial management of a trauma patient with a potential spinal cord injury involves securing the airway, breathing, and circulation, with immediate spinal immobilization to reduce further injury to the spinal cord. Immobilization involves the use of a rigid cervical collar, a backboard for transport, and spinal precautions for transfers. Prompt transfer to a specialized center to expedite proper care is recommended[7]. Systemic hypotension, defined as a systolic blood pressure of <90 mm Hg, should be avoided at all times, even for a brief period, as it has been associated with poorer neurological outcomes[8]. In the patient with polytrauma and a cervical spinal cord injury, hypovolemia is often compounded by neurogenic shock (loss of vascular tone and bradycardia). Hypovolemic shock requires aggressive fluid resuscitation with crystalloids coupled with alpha-agonists (e.g., phenylephrine) or mixed alpha-beta agonists (e.g., norepinephrine) as an adjunctive treatment to reduce peripheral vasodilation and to optimize spinal cord perfusion[9].

After the patient has been stabilized, a thorough clinical examination should be documented with use of the

TABLE I Established Tendon Transfer Scheme Compared with a Possible Nerve Transfer and Tendon Transfer Scheme

ICSHT Classification	Most Distal Functioning Muscle	ASIA Classification	Functional Goals of Tendon Transfers	Established Paradigm	Functional Goals of Tendon and Nerve Transfers	Paradigm with Nerve Transfers		
						Plan A	Plan B	Plan C
0	Trapezius	Above C5	None	No options	Elbow flexion	Spinal accessory nerve-to-free gracilis nerve transfer for elbow flexion		
0	Biceps	C5	None	No options	Elbow extension, wrist extension	Spinal accessory nerve-to-free gracilis nerve transfer for elbow extension	Long thoracic nerve or lateral pectoral nerve-to-free gracilis nerve transfer for elbow flexion and wrist extension	
0	Brachialis	C5	Elbow extension	Biceps-to-triceps tendon transfer if elbow flexion strength is 5/5	Elbow extension, wrist extension	Posterior deltoid nerve branch-to-triceps nerve transfer within 3-6 months	If triceps function after 12 months, and elbow flexion strength is 5/5, then brachialis-to-extensor carpi radialis brevis nerve transfer	If no triceps function after 12 months, and elbow flexion strength is 5/5, then biceps-to-triceps tendon transfer
1	Brachioradialis	C6	Elbow extension, wrist extension, passive pinch	Biceps-to-triceps tendon transfer and brachioradialis-to-extensor carpi radialis brevis tendon transfer, flexor pollicis longus tenodesis	Elbow extension, wrist extension, active pinch and grasp	Posterior deltoid nerve branch-to-triceps and supinator-to-anterior interosseous nerve transfer, and brachioradialis-to-extensor carpi radialis brevis tendon transfer	If posterior deltoid nerve branch-to-triceps nerve transfer insufficient, then biceps-to-triceps tendon transfer	If no pinch after 12 months, then flexor pollicis longus tenodesis
2	Extensor carpi radialis longus	C6	Elbow extension, active pinch	Biceps-to-triceps tendon transfer and brachioradialis-to-flexor pollicis longus tendon transfer	Elbow extension, active pinch and grasp, thumb extension	Posterior deltoid nerve branch-to-triceps and supinator-to-anterior interosseous nerve transfer	If pinch after 12 months, then brachioradialis-to-extensor pollicis longus tendon transfer, and if posterior deltoid nerve branch-to-triceps nerve transfer insufficient, then biceps-to-triceps tendon transfer	If no pinch after 12 months, then brachioradialis-to-flexor pollicis longus tendon transfer
3	Extensor carpi radialis brevis	C6/C7	Elbow extension, active pinch and grasp	Biceps-to-triceps tendon transfer, brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer	Elbow extension, active pinch and grasp, thumb and finger extension	Posterior deltoid nerve branch-to-triceps and supinator-to-posterior interosseous nerve transfer, brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer	If posterior deltoid nerve branch-to-triceps nerve transfer insufficient, then biceps-to-triceps tendon transfer	

continued

TABLE I (*continued*)

ICSHT Classification	Most Distal Functioning Muscle	ASIA Classification	Functional Goals of Tendon Transfers	Established Paradigm	Functional Goals of Tendon and Nerve Transfers	Paradigm with Nerve Transfers		
						Plan A	Plan B	Plan C
4	Pronator teres	C7	Elbow extension, active pinch and grasp	Biceps-to-triceps tendon transfer, brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer	Active pinch and grasp, thumb and finger extension	Posterior deltoid nerve branch-to-triceps and supinator-to-posterior interosseous nerve transfer, brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer	If posterior deltoid nerve branch-to-triceps nerve transfer insufficient, then biceps-to-triceps tendon transfer	
5	Flexor carpi radialis	C7	Active pinch and grasp	Brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer	Elbow extension, active pinch and grasp, thumb and finger extension	Supinator-to-posterior interosseous nerve transfer, brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer		
6	Extensor digitorum communis	C7	Active pinch and grasp	Brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer				
7	Extensor pollicis longus	C7	Active pinch and grasp	Brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer				
8	Flexor digitorum superficialis	C7	Active pinch and grasp	Brachioradialis-to-flexor pollicis longus tendon transfer, extensor carpi radialis longus-to-flexor digitorum profundus tendon transfer				
9	Flexor digitorum profundus	C8	Active pinch, thumb opposition	Flexor digitorum superficialis oppositionplasty, intrinsicplasty				
X	Variable							

ASIA International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI). This baseline evaluation is vital to establish the level of neurological injury, to guide treatment selection, and to monitor potential recovery and prognosis[8]. Computed tomography (CT) is the recommended imaging modality as standard radiographs have been shown to miss up to 6% of injuries[10]. In the setting of confirmed spinal cord injury, the entire thoracolumbar spine also must be evaluated with imaging studies as the incidence of noncontiguous spinal injuries is 5% to 10%[11]. Urgent magnetic resonance imaging (MRI) is recommended but is not mandatory in the immediate work-up unless there is an unexplained neurological deficit, in which case it is necessary to evaluate for cord compression or missed ligamentous injuries[12]. However, initial MRI has shown promising prognostic value for the evaluation of spinal cord injury[13].

The patient should be transferred to a critical care unit to provide continuous respiratory and hemodynamic support[8]. Life or limb-threatening injuries should be treated expeditiously by the appropriate teams while maintaining strict spinal immobilization. A concerted interdisciplinary effort is critical and can positively affect long-term outcomes[14].

Spinal cord ischemia is propagated by damage to the tenuous microvascular circulation of the spinal cord. Ischemia is a result of mechanical compression from the initial injury as well as localized progressive edema and hemorrhage. Early surgical decompression is recommended to reduce the secondary cascade of this ischemic-hypoxic injury[14]. The Surgical Timing in Acute Spinal Cord Injury Study (STASCIS) evaluated 313 patients with acute cervical spinal cord injury[15]. The study demonstrated that patients who underwent decompression ≤24 hours after the injury had 2.8 times greater odds of achieving at least a 2-grade improvement on the AIS by 6 months after the injury compared with those who underwent decompression >24 hours after the initial injury. There was also a trend toward a reduction in the rate of acute in-hospital complications. A subsequent multicenter prospective study in Canada involving 84 patients with spinal cord injury demonstrated similar benefits of early versus late decompression[16]. A secondary observational study demonstrated a significantly decreased length of hospital stay in patients classified as AIS A (complete injury) or AIS B (complete motor injury, incomplete sensory injury) who underwent decompression ≤24 hours after spinal cord injury compared with those who underwent decompression >24 hours after the injury ($p < 0.03$)[17]. Collectively, those studies helped to promote the concept of "Time is Spine," which highlights the importance of early diagnosis and treatment of acute spinal cord injury[12].

As discussed above, localized edema and microvascular trauma contribute to spinal cord ischemia and potentiate the secondary injury cascade. As a means to optimize spinal cord perfusion, the current guidelines from the American Association of Neurological Surgeons (AANS) and Congress of Neurological Surgeons (CNS) recommend maintaining the mean arterial pressure of at least 85 to 90 mm Hg for 7 days after an injury. While that recommendation is based on Level-III evidence, this method of blood-pressure augmentation has been shown to improve long-term outcomes as reflected by the AIS grade[18]. This method typically requires an arterial line for invasive blood pressure monitoring and a central venous catheter for the administration of vasopressors. Additional studies are underway to more closely evaluate the duration and level of blood-pressure augmentation that is required to maximize patient outcomes.

The administration of systemic corticosteroids has shown promising results in animal spinal cord injury models, prompting the National Acute Spinal Cord Injury Study (NASCIS) series in the 1980s to 1990s[19-21]. Subgroup analysis revealed a potential neurological benefit of high-dose methylprednisolone if given within 8 hours after the injury. However, serious adverse events that were noted in association with the methylprednisolone protocol, including pneumonia and sepsis, were thought to outweigh the potential neurological benefit. The methodology and analysis of those studies have been extensively debated. The 2013 AANS/CNS guidelines for the management of acute spinal cord injury did not recommend methylprednisolone[22]. However, a Cochrane review that evaluated 6 large studies on the use of methylprednisolone for patients with acute spinal cord injury demonstrated an overall 4-point increase in the ASIA motor score when methylprednisolone was administered within 8 hours after the injury[23]. That finding prompted the AO Spine 2017 guidelines to suggest that the administration of intravenous methylprednisolone for 24 hours should be considered if it can be started within 8 hours after the injury for patients with cervical spinal cord injury who do not have any medical contraindications[24,25].

Acute and Long-Term Rehabilitation

In the acute setting, the focus is on caring for the patient's concomitant injuries, preventing secondary complications, and restoring as much neurological function as possible. The goals of acute rehabilitation can range from primarily family and patient education (i.e., for patients with a high cervical complete injury) to complete functional independence (i.e., for patients with a low thoracic injury). At the head of the team is the physiatrist, who oversees the medical care of the patient as well as the overall rehabilitation team. An interdisciplinary team approach is used to optimize care, continuity, and outcomes. This team includes personnel from nursing, physical therapy, occupational therapy, speech therapy, psychology, nutritional care, and discharge planning (e.g., a medical social worker and/or

nurse care coordinator). Children and adolescents also receive additional support in the forms of art and music therapy, recreational therapy, child life therapy, orthotics, and hospital-based schooling. Appropriate consults are made to manage wounds, fractures, neurogenic bladder, and emotional distress as well as to introduce the patient and the family to future care options. Whenever possible, friends and relatives should be included in the care of the patient to cement the support network that the patient is likely to require in the long term. Typically, a primary and secondary caregiver are chosen by the patient and family to learn specific care goals in order to assist with a smooth transition home. Caregivers receive spinal cord injury education and hands-on training. Depending on the patient's functional level and home setup at the time of discharge, he or she may also benefit from skilled nursing and home health services.

Until the spine is deemed stable by the spine surgeon, therapy consists primarily of passive joint mobilization, limb positioning and/or splinting, and the prevention of skin pressure injury. After spine precautions are formalized and communicated with the rehabilitation team, progression of mobilization begins. Bed mobility is assessed, and then progress is made toward sitting. Because of deconditioning and autonomic impairment, patients are closely monitored for orthostatic hypotension and syncope. Bilateral thigh-high compression stockings and an abdominal binder help to prevent orthostatic hypotension, but oral medications are sometimes needed during the waking hours, especially when the patient is out of bed. Patients progress from sitting up in bed to sitting up in a manual or power wheelchair with appropriate pressure-relieving cushions. Pressure mapping is completed at the beginning of the rehabilitation stay to ensure safe pressures over osseous prominences and the prevention of skin breakdown. Once sitting is tolerated and spine precautions are reviewed, a standing program is initiated with use of a tilt table followed by progression to a standing frame or a standing wheelchair. Depending on the patient's medical stability, functional level, and level of injury, the patient progresses through a customized rehabilitation program, which may include locomotor training, neuromuscular electrical stimulation, functional electrical stimulation, cycling, aerobic exercise, strengthening, passive and active range of motion, and optimization of communication (i.e., using a speaking valve for patients with high cervical injury who are ventilator-dependent). Patients with tetraplegia are screened for future upper-limb interventions to optimize function. Occupational therapy plays an extremely important role in this screening process.

After discharge from acute rehabilitation, ongoing goals are achieved through outpatient therapy and a daily home exercise program. Long-term rehabilitation goals include mitigating complications, reducing pain and spasticity, and maximizing independence. These goals can be achieved with a variety of assistive devices, mobility aids, and home and vehicle modifications.

Medical management during inpatient rehabilitation includes progression of respiratory care, prevention of orthostatic hypotension and autonomic dysreflexia (for lesions at T6 and above), development of neurogenic bowel and bladder programs, minimization of decubitus ulcers, and optimization of nutrition. The bowel and bladder program must be practical and optimized for each individual's school, recreation, and work schedule.

Surgical Options for Improving Ventilation

For most patients with high cervical spinal cord injuries, the restoration of upper limb function is not possible with nerve or tendon transfers. However, it is possible to wean many of these patients off a ventilator during the day. Negative-pressure ventilation, even for part of the day, can mitigate the long-term risks of positive-pressure ventilation[26,27]. If the phrenic nerve lower motor neurons are

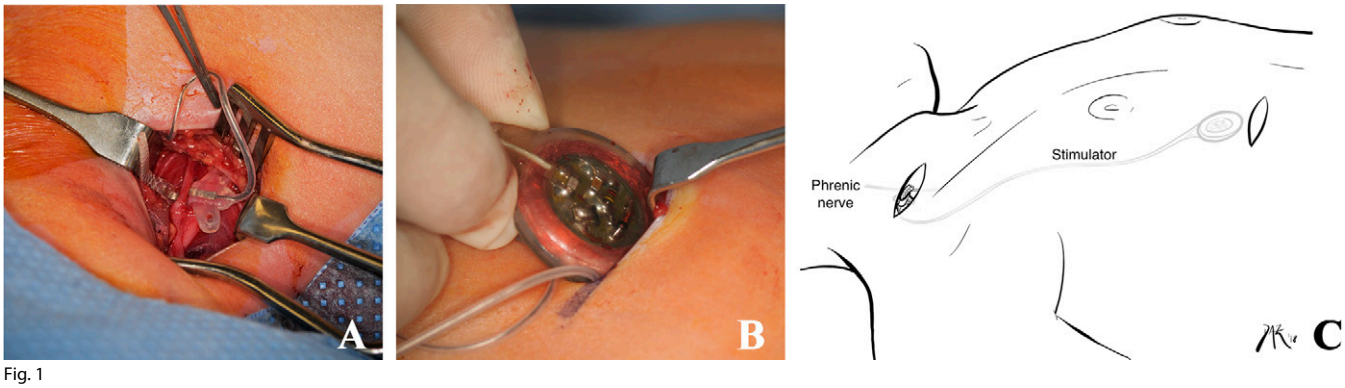

Fig. 1

Figs. 1-A, 1-B, and 1-C Photographs and illustration showing the placement of a phrenic nerve pacer (Avery Laboratories I-110A) via a cervical approach in a patient with a stimulatable phrenic nerve. A supraclavicular incision is made and the phrenic nerve is identified deep to the carotid sheath on the anterior scalene muscle. The electrode is placed around the nerve (**Fig. 1-A**), the receiver is inserted through a separate thoracic incision (**Fig. 1-B**), and the cables are tunneled below the skin (**Fig. 1-C**). (**Figures 1-A and 1-B** are reproduced with permission from Shriners Hospitals for Children – Philadelphia. **Figure 1-C** is reproduced with permission from Dan A. Zlotolow, MD.)

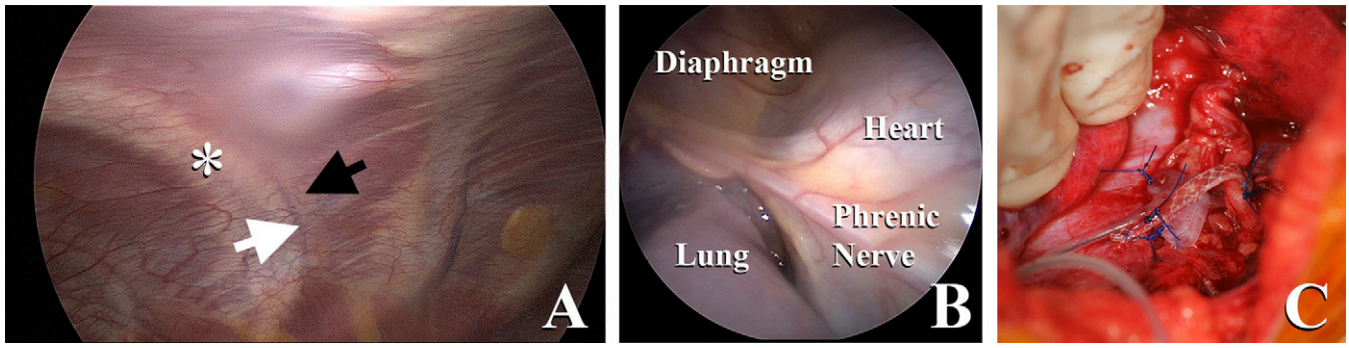

Fig. 2

Figs. 2-A, 2-B, and 2-C Arthroscopic images and intraoperative photograph made during intercostal-to-phrenic nerve transfer via a thoracoscopic approach. **Figs. 2-A and 2-B** The intercostal nerves (black arrow), intercostal vascular bundle (white arrow), ribs (white star) (**Fig. 2-A**) and phrenic nerve (**Fig. 2-B**) can be easily identified and dissected free. **Fig. 2-C** The pacer is applied to the intercostal nerve at the inflection point of the intercostal nerve from the chest wall. (Reproduced with permission from Shriners Hospitals for Children – Philadelphia.)

intact (below the zone of injury) but are not functional because they are disconnected from the upper motor neurons, either the phrenic nerve or the diaphragm can be paced with use of a buried (Avery Laboratories I-110A) or transcutaneous pacer (NeuRx RA/4 system, Synapse Biomedical), respectively. Because the lower motor neurons are intact, pacers can be implanted many years after injury with good results[28]. We prefer to place the phrenic nerve pacer via a transverse supraclavicular incision (Fig. 1).

If the lower motor neurons to the phrenic nerve are within the zone of injury, the diaphragm is denervated and will undergo irreversible end-plate demise as early as 18 months after the injury. The diaphragm must be reinnervated within 12 months after the injury with a nerve transfer, typically from the intercostal nerves, in order for either method of pacing to work[29]. In our experience, we have seen well-intentioned surgeons place a diaphragmatic pacer into a denervated diaphragm, only to watch it stop functioning as the muscle slowly loses it contractile ability between 18 and 24 months after injury.

Evaluation of phrenic nerve function requires stimulation of the brachial plexus at Erb's point while visualizing the diaphragm with either ultrasound or fluoroscopy. Nerve stimulation must be done at least 3 weeks after the injury to allow for Wallerian degeneration to occur and preferably should be done between 6 and 9 months to allow for spontaneous recovery.

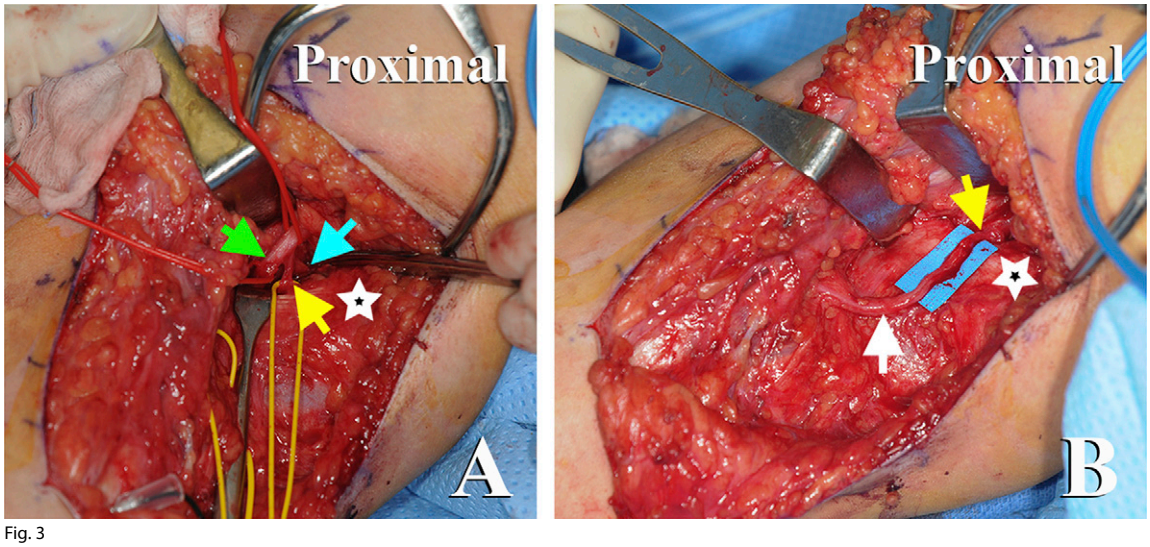

Fig. 3

Figs. 3-A and 3-B Photographs made during transfer of the posterior deltoid branch of the axillary nerve to the common nerve branch of the triceps nerve through an axillary approach. **Fig. 3-A** The axillary nerve can be found on the medial-superior edge of the latissimus dorsi tendon (white star) near its insertion point. The axillary nerve has 3 distinct branches at this level: the anterior deltoid (green arrow), posterior deltoid and cutaneous (yellow arrow), and recurrent teres minor branches (blue arrow). **Fig. 3-B** The posterior deltoid branch (yellow arrow) can be coapted to the common branch of the triceps (white arrow) with no tension with use of fibrin glue. (Reproduced with permission from Shriners Hospitals for Children – Philadelphia.)

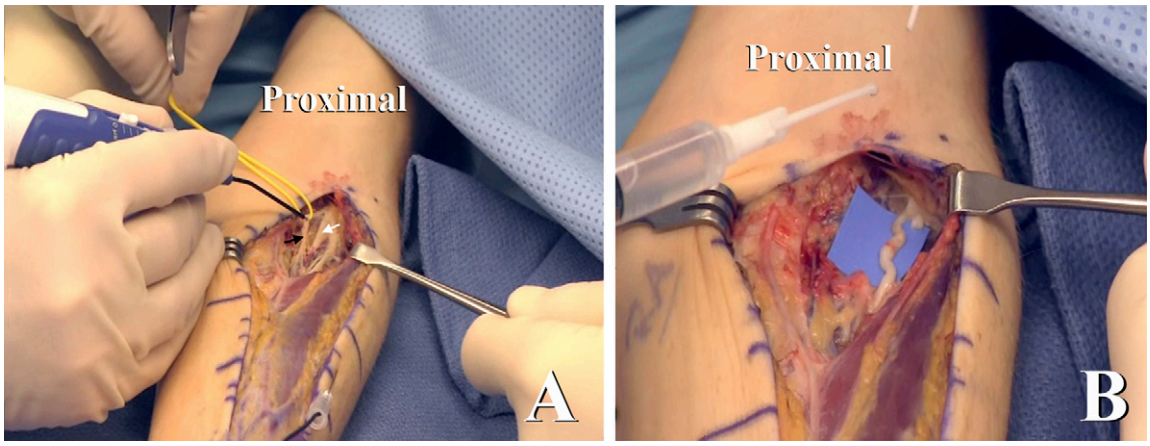

Fig. 4

Figs. 4-A and 4-B Photographs made during supinator-to-posterior interosseous nerve (SPIN) transfer through an anterior forearm approach just lateral to the biceps tendon. **Fig. 4-A** The supinator has 2 major branches that branch from and bracket the posterior interosseous nerve (white arrow), although the more medial branch is often dominant (black arrow). **Fig. 4-B** The coaptation can be routinely accomplished free of tension with use of fibrin glue. (Reproduced with permission from Shriners Hospitals for Children – Philadelphia.)

There is no standard for how many intercostal nerves to transfer[30], but we have had success with transferring only 1 nerve with both motor and sensory branches. Our preference is to perform the nerve transfer thoracoscopically (Fig. 2), which for us requires the assistance of a thoracic or general surgeon who is facile with thoracoscopy. The phrenic pacer is placed through a small thoracotomy incision on the chest wall at the inflection point of the intercostal nerve from the chest wall. A diaphragmatic pacer can be inserted laparoscopically with use of the same setup and scoping equipment.

Pacing can begin as soon as the wounds are healed and the nerve transfers are no longer at risk of displacement, typically around 3 weeks postoperatively. If no nerve transfer was required, stimulation of either the phrenic nerve or the diaphragm is ramped up from a few minutes a few times a day to all day over the course of 12 weeks. The aim is to give the diaphragm a chance to hypertrophy and increase in strength and endurance without overwhelming the muscle. If a phrenic nerve pacer is applied proximal to the nerve transfer site, we recommend a similar start for the stimulation but delay ramping up the time that the stimulator is active until

TABLE II An Incomplete List of Possible Nerve and Tendon Transfers

Transfer	Innervation	Loss	Gain
Posterior deltoid to triceps nerve transfer	C5	Weakened shoulder extension	Elbow extension
Posterior deltoid to triceps tendon transfer	C5	Weakened shoulder extension	Elbow extension
Biceps to triceps tendon transfer	C5/C6	Weakened elbow flexion/ supination	Elbow extension
Brachialis to anterior interosseous nerve transfer	C6	Weakened elbow flexion	Digital flexion
Supinator to anterior interosseous nerve transfer	C6	Weakened supination	Digital flexion
Supinator to posterior interosseous nerve transfer	C6	Weakened supination	Digital extension
Brachioradialis to flexor pollicis longus tendon transfer	C6	None	Thumb flexion
Extensor carpi radialis longus/extensor carpi radialis brevis to flexor digitorum profundus tendon transfer	C6/C7	Weakened wrist extension	Finger flexion
Pronator teres to anterior interosseous nerve transfer	C7	Weakened pronation	Digital flexion
Pronator teres to flexor pollicis longus tendon transfer	C7	Weakened pronation	Thumb flexion
Pronator teres to flexor digitorum profundus tendon transfer	C7	Weakened pronation	Finger flexion
Flexor carpi radialis to extensor digitorum communis tendon transfer	C7	Weakened wrist flexion	Finger extension
Palmaris to abductor pollicis brevis	C7	None	Thumb abduction
Extensor carpi ulnaris to abductor pollicis brevis	C7	Weakened wrist ulnar deviation	Thumb abduction/ opposition

TABLE III Comparison of Nerve and Tendon Transfers

	Nerve Transfer	Tendon Transfer
Strength	Good	Better
Dexterity/control	Better	Good
Success rate	Good	Better
Technical difficulty	Advanced	Moderate
Rehabilitation	Delayed	Demanding
Timing	6-12 months after injury	Anytime

the diaphragm begins to contract. Stimulation of the nerve may improve the success of the nerve transfer[31]. For the insertion of a diaphragm pacer after nerve transfer, to our knowledge, there is no evidence to support any specific protocol.

Surgical Options for Improving Limb Function

Despite an incidence of >5,000 new tetraplegia cases per year, with >65% meeting the indications for surgery[6], <500 procedures are performed annually to improve upper limb function. Tendon transfers have been shown to improve quality of life and function, yet spinal cord rehabilitation facilities are often either wary of referring patients to one of the few centers that specialize in tetraplegia management or are unaware that they exist[6]. As a result, patients have been sentenced to unnecessary levels of dependency. Patients can typically gain 1 cervical spinal level of function through tendon transfers[3]. Most patients with injuries at or caudad to the C6 level can become independent through tendon and nerve transfers.

This paucity of referrals has become even more frustrating as the last few years have brought forth new, but time-sensitive, options for restoring upper-limb function to patients with tetraplegia. The established paradigm of tendon transfers is being augmented with nerve transfers, often allowing restoration of 2 cervical spinal cord levels of function[32-34]. However, because nerve transfers require a viable muscle target for reinnervation, and irreversible motor end-plate demise occurs as early as 18 months after an injury, most nerve transfers have limited success if performed >12 months after the injury[35]. Options for nerve transfer include posterior deltoid to triceps (Fig. 3), brachialis to anterior interosseous nerve, supinator to posterior interosseous nerve (Fig. 4), supinator to anterior interosseous nerve, supinator to extensor carpi radialis brevis, and pronator teres to anterior interosseous nerve (Table II), but any expendable motor nerve can be considered as a donor nerve.

While nerve transfers have ushered in a revolution in tetraplegia management, they have brought confusion to a well-established treatment algorithm. We favor a staged approach that tries to maximize the benefits of both nerve and tendon transfers (Table III) while retaining the established ladder of tetraplegia reconstruction (Table I)[3]. Our strategy is by no means an established algorithm and is subject to change on an individual basis, but it does serve as a guide for integrating nerve and tendon transfers.

We perform our procedures with 2 teams working simultaneously to minimize anesthesia time. Often, we bias our reconstruction toward tendon transfers in 1 arm for strength and toward nerve transfers in the other arm for dexterity. The dominant side is often chosen for tendon transfers in order to preserve the supinator for self-feeding in case a biceps-to-triceps tendon transfer (Fig. 5) is required to augment a posterior deltoid-to-triceps nerve transfer[36].

If only nerve transfers were performed, we wait until the patient shows strength of at least 1/5 in the reinnervated muscles before initiating rehabilitation. If combined nerve and tendon transfers were performed, we typically wait 2 weeks for the wounds to heal before initiating rehabilitation. If only tendon transfers were performed, rehabilitation can begin within a week after surgery, provided that the donor and recipient tendons allowed for good fixation. Children who have been injured at a young age can have diminutive tendons even into adulthood and require gentle rehabilitation delayed at least 3 weeks.

Rehabilitation protocols vary for the specific blend of procedures performed but typically require twice-daily sessions with a therapist for a minimum of 6 weeks. Lack of compliance with the rehabilitation protocol is the primary mode of failure after surgery. We have been using botulinum toxin A to weaken the biceps muscle after biceps-to-triceps tendon transfers, resulting in a nearly 50% reduction in the failure rate of this transfer. Biofeedback and mirror therapy may be helpful as well.

Fig. 5

Photograph of a patient with a C6-level spinal cord injury, made 1 year after bilateral biceps-to-triceps tendon transfers and tendon transfers for pinch and grasp. After surgery, the patient became able to self-transfer, push his wheelchair up ramps, self-catheterize, and self-feed. (Reproduced with permission from Shriners Hospitals for Children – Philadelphia.)

Overview

Spinal cord injuries are often devastating neurological injuries that impact every aspect of a patient's life. Without modern medical care, many of these patients would die as a result of complications within the first few years after the injury. However, current management for the majority of patients has not yet caught up to advances in what is technically possible. This gap between the care that is given and the care that is available is one of the great tragedies of the American medical system. In countries with more coordinated health care delivery systems, patients are routed reliably to centers of excellence that follow accepted clinical guidelines based on the best available evidence, experience, and cost efficiency.

As the population of patients with spinal cord injuries expands, the deployment of advances in technology and surgical techniques to this population will become even more critical. We must work to remove the existing roadblocks to care for what we can do today, so that the advances of tomorrow can benefit these patients as soon as they become available.

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